- *All letters must be typed with double spacing and signed by all authors.*
- *No letter should be more than 400 words.*
- *For letters on scientific subjects we normally reserve our correspondence columns for those relating to issues discussed recently (within six weeks) in the BMJ.*
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Milk, butter, and heart disease

SIR,—The MRC epidemiology unit in Wales has attracted a considerable amount of media attention as a result of reports that people who drink milk are less prone to heart attacks than those who do not and that butter eaters experience fewer heart attacks than those using polyunsaturated margarines.[1] This information has not been formally presented in the medical journals, but it seems important to discuss the findings that are available in the report published and distributed by the unit.[2]

Milk in Caerphilly—The dietary data presented are from 2818 middle aged men (45-59 years at screening) from Caerphilly and five adjacent villages. The men were asked, "How much milk (not skimmed or dried) do you drink per day in tea or coffee, in milky drinks, with cereals?" The categories for choice were "none, half pint or less, half to 1 pint, more than 1 pint." Table I shows the number of men and the number (and percentage) of men in each category experiencing a major ischaemic heart disease event in 5 years. The report states that "the relationship with ischaemic heart disease is strong and adjustment in a regression model for a number of co-variates reduces the trend only marginally, even when prevalent ischaemic heart disease at base-line is added." Media attention has focused on the more than eight-fold difference in heart attack rate seen between non-drinkers of milk and those drinking more than one pint, in the data presented, which is not standardised for any other variables.

Butter in Caerphilly—The men were asked what they usually ate on bread—butter, soft margarine (specifying brand), or hard margarine. Table II shows that those using polyunsaturated margarine had 1·9 times the heart attack rate seen in butter eaters. The report states that the relation remained significant even after possible confounding factors, including evidence of ischaemic heart disease at baseline, had been allowed for. However, only the unadjusted data are presented. No information is provided as to which possible confounding variables were adjusted for in the regression models, apart from ischaemic heart disease at baseline.

British regional heart study—In view of the considerable publicity given to these data we have examined similar information obtained from 7735 middle aged men (40-59 years at screening) in the British regional heart study, based on general practices in 24 towns in England, Wales, and Scotland and representative, in socioeconomic terms, of the middle aged male population of Great Britain.[3] The men were asked simply whether they used milk on cereals, in tea, in coffee, or as a milk drink. They were also asked what kind of spreading fats they used at home: butter or margarine (specifying brand). The men have been followed up for both fatal and non-fatal cardiovascular events[4] and the data presented relate to a follow up of 9·5 years in all men.

Milk intake—As no quantitative information was obtained, the men have been grouped rather than ranked, although there is some rough quantitative quality to the grouping (table III). The first two categories are clearly drinking less milk than the last two categories, who take milk with cereal as well as other milk contributions. The relation between the higher milk intake and cereal intake is of interest, as a relation has been shown between cereals (fibre) and coronary heart disease.[5]

There is a decrease in the incidence of heart attack as milk intake increases, similar in magnitude and direction to that seen in the MRC data for their first three groups (table I). Examination of the characteristics of men in the several milk drinking categories shows that non-drinkers and heavier drinkers of milk have many clear differences (table IV). The non-drinkers and those who drink little milk are significantly older and include a greater proportion of manual workers, a higher percentage of current cigarette smokers, more obese men, men with higher systolic blood pressure, a higher proportion of inactive men, and a lower proportion of men taking vigorous physical activity. Most striking is the recall of a doctor's diagnosis of ischaemic heart disease at screening: 13·3% in the non-drinkers of milk and 3·1% in the heaviest milk drinkers.

Clearly these findings must be taken into account if we are to interpret the relation of milk to ischaemic heart disease. Simple exclusion of men with recall of a doctor's diagnosis of ischaemic heart disease does not noticeably alter the trend of the relation seen in table III. Other variables may be taken into account by using a multiple regression model incorporating age, social class, cigarette smoking, serum total cholesterol concentration, systolic blood pressure, physical activity index, and recall of a doctor's diagnosis of ischaemic heart disease and diabetes. Table III also shows the relative risk of a heart attack in the several milk drinking categories, with those in the heaviest intake category serving as a baseline group. The higher rate of heart attack in the non-drinkers of milk seems to be largely accounted for by the well recognised risk factors for ischaemic heart disease.

Butter—Because of the simplicity of our questionnaire, the men were categorised by their usual habit (table V). In the univariate analysis, there was a significant difference between the type of fat spread and the risk of heart attack, with butter eaters having lower rates than non-butter eaters (margarine only or no spread).

Once again, it is important to examine the characteristics of the butter eaters and non-butter eaters to determine which factors might account for the observed difference in heart attack rates. There was little difference in age, social class, cigarette smoking, systolic blood pressure, or blood cholesterol patterns between the groups (data not presented). However, 28% of men taking no fat spread were obese (body mass index ≥ 28 kg/m^2) compared with 18% of butter eaters. The non-butter eaters (no spread or margarine only) had a higher prevalence of recall of a doctor's

TABLE I—*Milk intake and major ischaemic heart disease events (unadjusted) over five years in men in Caerphilly*

Daily intake of milk	No of men	No (%) with major ischaemic heart disease event
None	162	16 (9·9)
≤½ pint	1104	70 (6·3)
<1 pint	973	56 (5·8)
≥1 pint	164	2 (1·2)

TABLE II—*Butter and margarine use and major ischaemic heart disease events over five years in men in Caerphilly*

Fat spread	No of men	No (%) with major ischaemic heart disease event
Butter	1380	73 (5·3%)
Butter and margarine	208	15 (7·2%)
Polyunsat margarine	250	24 (9·6%)
Other soft margarine	416	30 (7·2%)
Hard margarine	141	6 (4·3%)

TABLE III—*British regional heart study. Milk intake and major ischaemic heart disease events over 9·5 years' follow up*

Milk intake	No of men	No (%) of patients with ischaemic heart disease	Relative risk of heart attack*		
			Unadjusted	Adjusted†	Adjusted‡
None	241	24 (9·9%)	1·66	1·43	1·13
Tea or coffee only	2361	219 (9·3%)	1·54	1·23	1·12
Drink	1653	132 (7·9%)	1·29	1·11	1·06
In cereal	1840	131 (7·2%)	1·17	1·11	1·00
In cereal plus drink	1634	102 (6·2%)	1·00	1·00	1·00

*Over 9·5 years.
†Adjusted for age, social class, smoking, blood cholesterol concentration, and systolic blood pressure.
‡Adjusted also for ischaemic heart disease and diabetes screening.

TABLE IV—*British regional heart study. Characteristics at screening of men in different categories of milk intake*

Milk intake	Age (years)	Manual workers	Current smokers (%)	Obese* (%)	Serum total cholesterol (mmol/l)	Systolic blood pressure (mm Hg)	Level of activity		Recall doctors' diagnosis		Drinking	
							Inactive (%)	Vigorous (%)	Ischaemic heart disease (%)	Diabetes (%)	Non-drinkers (%)	Heavy drinkers (%)
None	50·8	59	38	27	6·36	146·0	13	20	13	2·1	11	16
Tea or coffee only	50·7	67	50	22	6·32	146·9	11	18	7	1·7	5	15
Drink	50·4	71	49	22	6·31	146·4	11	20	4	1·0	6	16
In cereal	50·0	46	29	16	6·25	143·9	7	24	7	1·5	6	6
In cereal plus drink	49·7	51	36	16	6·30	142·7	6	25	3	1·8	7	5

*Body mass index ≥28·0 kg/m².

TABLE V—*British regional heart study. Fat spread usually used and major ischaemic heart disease events over 9·5 years of follow up*

Fat spread	No of men	No (%) with ischaemic heart disease	Relative risk (95% confidence interval) of heart attack*
None	136	11 (8·1)	0·57 (0·22 to 1·46)
Margarine only	2735	247 (9·0)	1·00
Margarine and butter	1038	66 (6·4)	0·76 (0·55 to 1·04)
Butter only	3808	283 (7·4)	0·87 (0·79 to 1·06)

*After exclusion of men with recall of doctor's diagnosis of ischaemic heart disease at screening; adjusted for age, social class, cigarette smoking, and blood cholesterol concentration.

diagnosis of ischaemic heart disease than men using butter (10·1% *v* 4·2%). These findings strongly suggest that pre-existing illness (obesity and heart disease) is associated with choice of fat spread.

After exclusion of the men with recall of a doctor's diagnosis of ischaemic heart disease and adjustment for other key risk factors no significant association was seen between butter intake and heart attack rates. There seems to be no evidence that butter eating is protective against ischaemic heart disease.

Conclusions—Data from the British regional heart study show that men who had the highest milk intake at initial screening had a lower rate of heart attack than men who drank no milk. Similarly, men who used only butter as a spread at initial screening had a lower rate of heart attack than men who used margarine only or no spread at all. However, the characterstics of the men in the several milk drinking or fat spread categories are very different, and these differences must be taken into account when assessing the importance of the association in terms of possible causality. When these background characteristics are taken into account we can find no significant association between milk intake or fat spread use and the incidence of heart attack in these middle aged British men.

People with disease often change their lifestyle and this must be fully considered when attempting to associate any specific behaviour with a disease end point and to interpret the findings in terms of causality. Men in the United Kingdom who do not drink milk at all or do not use butter are small groups who differ in many ways from the rest of the population. Comparisons between extreme groups may yield large relative risks, but caution is required before causality is invoked.

A G SHAPER
GOYA WANNAMETHEE
MARY WALKER

Department of Public Health and Primary Care,
Royal Free Hospital School of Medicine,
London NW3 2PF

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Thrombolytic treatment for recurrent myocardial infarction

SIR,—Further to Dr Harvey White's editorial regarding thrombolytic treatment for recurrent myocardial infarction,[1] we have recently completed a postal survey of the age related admission and thrombolysis policies of the 175 coronary care units in the United Kingdom identified from the *Directory of Emergency and Special Care Units 1990*.[2] As part of that survey we inquired into the use of thrombolytic drugs for recurrent myocardial infarction.

Of the 175 questionnaires, 134 were returned. Recombinant tissue plasminogen activator was the agent most commonly given (by 123 units) to patients who had received previous treatment for thrombolysis. Three units used anistreplase as the sole alternative to streptokinase. Eight units had no alternative thrombolytic drug to streptokinase, one of which operated a policy of giving double the standard dose of streptokinase for reinfarction.

Fifty seven of the 123 units using recombinant tissue plasminogen activator indicated a time policy for when streptokinase would not be given for the treatment of reinfarction. A total of 18 different policies were operated by these units. Two units gave recombinant tissue plasminogen activator to all patients who had previously received streptokinase, no matter the time interval since the initial dose. One unit gave streptokinase up to five days after the initial treatment but from then on only recombinant tissue plasminogen activator. Of the remaining 16 policies, the two most common were to give recombinant tissue plasminogen activator if streptokinase had been administered in the previous 12 months (23 units) or previous six months (14 units).

Clearly, at present there is no consensus on the most appropriate management for recurrent myocardial infarction other than to give recombinant tissue plasminogen activator if available. Some of the policies currently used are likely to provide ineffectual treatment for many patients.

If streptokinase is to be used for treating recurrent infarction it may be worth considering the use of skin testing to try to identify those patients at risk of developing anaphylaxis. A previous small study using 100 IU streptokinase intradermally showed this to be a sensitive and specific indicator of raised concentrations of IgE to streptokinase.[3] The test takes only 15 minutes so would not cause a great delay to starting treatment. After a negative test result higher doses of streptokinase could be given with more confidence of not precipitating a major anaphylactic reaction, although the possibility of later allergic reactions of IgG to streptokinase would still remain.

N J DUDLEY
E BURNS

Department of Medicine for the Elderly,
St James's University Hospital,
Leeds LS9 7TF

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Patients with chest pain in accident and emergency departments

SIR,—The article by Mr S S Tachakra and colleagues[1] contains a number of flaws that could lead others to adopt dangerous practices in patients who present with possible myocardial infarction.

Firstly, there is no record, other than stating the number referred by their general practitioners, of the presentation. There is a world of difference between patients who drop in to an accident department with chest pain that has troubled them for a few days and those who experience a severe enough pain to cause them to dial 999 for an ambulance in the belief, usually correct, that they are experiencing a heart attack.

Secondly, it is not safe to assume that the patients who did not respond to the postal survey had not had further trouble. An alternative explanation is that they had all died or been admitted to another hospital with myocardial infarction.

Thirdly, Mr Tachakra and colleagues should not have assumed that all the patients in whom the pain had settled down had not had a heart attack. Most patients with documented myocardial infarction experience pain for a number of hours, but many are free of pain by the next day.

Fourthly, there is an implication that it was sufficient to obtain an electrocardiogram to exclude myocardial infarction (or other serious cardiovascular disease such as aortic dissection). Standard medical textbooks emphasise that a normal tracing does not exclude myocardial infarction, particularly within the first few hours, and the patient with a typical history, particularly if it is supported by the presence of one or more major risk factors, must be assumed to have had or be in the process of developing a myocardial infarct. Indeed, many doctors do not wait for the development of changes on the electrocardiogram to start thrombolytic therapy.

Fifthly, Mr Tachakra and colleagues make no reference to the use of a short stay observation ward or area, which many doctors and accident staff think is the ideal way to manage patients with chest pain in whom the diagnosis is not immediately apparent.

The authors make an important point, although it is dismissed in one sentence. This concerns the training of accident and emergency staff—probably the most important single factor in avoiding unnecessary misdiagnosis. However, they give